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# The Activities of 1'-acetoxychavicol Acetate on SW620 Colorectal Cancer Cells Line

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## Abstract

Cancer is a significant cause of mortality worldwide, including in Thailand. However, chemotherapy has serious side effects. Ongoing research on the compound 1'-acetoxychavicol acetate (ACA) has revealed various medicinal properties, including anticancer and anti-inflammatory activities. Although ACA has been found to affect cancer cell lines through different mechanisms, few reports have focused on its effectiveness against colorectal cancer cell lines. This research aims to determine the anticancer activities of ACA on the SW620 cell line. Anticancer activities, including anti-proliferation, anti-migration, and anti-invasion, were evaluated using methylthiazolyldiphenyl-tetrazolium bromide (MTT), colony formation, scratch assays, invasion assays, and qRT-PCR. The results showed that ACA exhibited cytotoxic effects ($IC_{50}$ values) and anti-proliferative activity in a dose-dependent manner. ACA also demonstrated anti-migration and anti-invasion activities in a dose-dependent manner. Additionally, the qRT-PCR results showed that ACA significantly decreased Kirsten rat sarcoma viral oncogene homolog (KRAS) gene expressions compared to the control group. ACA exhibits anti-proliferative, anti-migratory, and anti-invasive activities in SW620 cells. These findings suggest the potential of ACA as a therapeutic agent and may provide insights that significantly advance our understanding of cancer biology and treatment.

***Keywords***: *colorectal cancer; 1'-acetoxychavicol acetate; anti-cancer; Kirsten rat sarcoma viral oncogene homolog; KRAS*

## 1. Introduction

Cancer is a non-communicable disease (NCD) that has become the leading cause of mortality ( Horton, 2013; Ray & Pareek, 2023). The progression of cancer, which can take 10-20 years and often has a poor prognosis, leads to metastasis to distant organs (Mitry et al., 2010). A primary etiological factor in the development of malignant tumors is the loss of function in the Kirsten rat sarcoma viral oncogene homolog (*KRAS*) gene, a critical regulator of cell proliferation. Disruption of *KRAS* function can lead to dysregulated cell proliferation, characterized by rapid and uncontrolled cell growth, ultimately contributing to tumorigenesis (Iacopetta, 2003; Lamouille et al., 2014). Cancer treatment typically encompasses surgical intervention, chemotherapy, radiation therapy, and targeted therapy, with the specific approach determined by the clinical context (Danaei et al., 2005). However, chemotherapy can have toxic effects on rapidly dividing cells, resulting in side

effects such as alopecia, nausea, vomiting, ulcers, mucositis, and drug resistance (Xu et al., 2020; Thiha & Sawasdipong, 2025). Therefore, addressing gaps or interruptions in cancer treatment regimens remains a significant challenge, necessitating improvements in current therapeutic strategies to optimize patient outcomes.

*Alpinia galanga* (L.) Willd produces secondary metabolites, particularly 1′-acetoxychavicol acetate (ACA), found in its fresh rhizomes. This plant is used for cooking and traditional medicine (Xu et al., 2008). Reported medicinal properties of ACA include anti-inflammatory, anti-dementia, antimicrobial, antidiabetic, and anticancer activities (In et al., 2012). In colorectal adenocarcinoma, ACA at an $IC_{50}$ of approximately 80 μM showed anti-proliferative activity in SW480 cells (Baradwaj et al., 2017). Previous research has demonstrated that ACA exhibits promising anticancer activity across various cancer types, often displaying synergistic effects with conventional chemotherapies in cervical carcinoma cells. Further studies elucidated ACA's mechanisms in breast cancer by inhibiting key oncogenic signaling pathways and cell cycle regulators, and in colorectal cancer by inducing apoptosis, cell cycle arrest, and DNA damage. Moreover, ACA has shown cytotoxic and pro-autophagic effects in lung cancer cells through a Beclin-1-independent pathway, indicating its potential to target diverse cellular mechanisms crucial to cancer progression (Nelson et al., 2020; Ketkomol et al., 2024). However, studies on the effects of ACA in colorectal cancer are still limited.

## 2. Objectives

This study aims to evaluate the effects of ACA on the SW620 colorectal cancer cell line.

## 3. Materials and Methods

### 3.1 Reagents and Materials

1′-Acetoxychavicol acetate (ACA) (College of Pharmacy, Rangsit University, Thailand), ethanol, methanol, dimethyl sulfoxide (DMSO), Roswell Park Memorial Institute-1640 (RPMI-1640) medium (Sigma, USA), fetal bovine serum (FBS) (Sigma, USA), Trypan blue dye (Sigma, USA), paraformaldehyde (Sigma, USA), methylthiazolyldiphenyl-tetrazolium bromide (MTT), crystal violet, Matrigel basement membrane (Corning, USA), and penicillin/ streptomycin (Sigma, USA).

### 3.2 Source of Activity Compound

1′-Acetoxychavicol acetate (ACA) was isolated from fresh *Alpinia galanga* (L.) rhizomes in November 2017. Furthermore, extraction and purification methods were developed and subsequently patented by Pradubyat and colleagues (Publication No. US 2002/0192262 A1) (Pradubyat et al., 2022).

### 3.3 Cytotoxicity Activity

The cytotoxic activity of ACA on the SW620 cell line was evaluated using the MTT assay (Jongrungruangchok et al., 2023). SW620 cells were exposed to ACA at concentrations ranging from 10 to 60 μM. For controls, 0.2% ethanol in RPMI complete medium served as the negative control, while cisplatin (Cis) was used as the positive control. Following treatment for 24 to 72 hours, MTT solution (0.45 mg/mL) was added to the cells. The resulting formazan crystals were dissolved in dimethyl sulfoxide (DMSO), and the relative number of viable cells was quantified by measuring absorbance at 570 nm.

### 3.4 Colony Formation Assay

To assess the long-term effects of ACA on cell proliferation, 2,000 SW620 cells were seeded into 6-well plates and treated with ACA at non-toxic concentrations determined from the MTT assay. The cells were incubated for 7 days to allow colony formation. After incubation, colonies were fixed with 3.7% paraformaldehyde for 20 minutes and subsequently stained with 0.1% crystal violet for 20 minutes to enable visualization and quantitative assessment of colony formation.

### 3.5 Migration Assay

To assess the effect of ACA on cell migration, a wound was created in a confluent monolayer of SW620 cells ($2 \times 10^5$) seeded in 6-well plates after overnight incubation, using a sterile 200 μL pipette tip. The cells were then washed with serum-free RPMI, and the medium was replaced with serum-free RPMI containing non-toxic concentrations of ACA. Cell migration into the wound area was monitored for 48 hours. Wound closure was quantified by comparing the wound area at 0 and 48 hours using ImageJ software (Version 1.45). The percentage of wound closure was calculated as the ratio of the area covered by migrated cells to the initial wound area.

### 3.6 Transwell Migration Assay

To assess the effect of ACA on cell migration and invasion, SW620 cells were sub-cultured at a density of $3 \times 10^4$ cells/mL in serum-free medium and seeded into the upper chambers of Matrigel-coated transwell inserts. The cells in the upper chambers were treated with non-toxic concentrations of ACA. After incubation, the cells were fixed with 3.7% paraformaldehyde and stained with 0.1% crystal violet for 15 minutes. Non-invading cells remaining in the upper chamber were carefully removed, and the cells that had invaded through the Matrigel to the lower surface of the membrane were counted using an inverted microscope.

### 3.7 Gene Expression Analysis

To investigate the inhibitory effects of ACA on the expression of genes associated with cell proliferation, quantitative real-time PCR (qRT-PCR) was performed. SW620 cells were cultured in 6-well plates and treated with the indicated concentrations of ACA. After a 24-hour incubation, total RNA was extracted from the cells using the Total RNA Mini Kit (Geneaid, Taiwan) according to the manufacturer's protocol. The extracted RNA was then reverse-transcribed into complementary DNA (cDNA) using the iScript™ Select cDNA Synthesis Kit. qRT-PCR was performed using SYBR Green chemistry, with the synthesized cDNA as the template and specific primer pairs for the *KRAS* gene. Glyceraldehyde 3-phosphate dehydrogenase (*GAPDH*) was used as the housekeeping/reference gene for normalization, and data analysis was conducted using the ΔΔCt method.

### 3.8 Statistical Analysis

All data are presented as the mean ± standard error of the mean (SEM). Statistical hypotheses were tested using one-way analysis of variance (ANOVA), followed by Tukey's Honestly Significant Difference ( HSD) post hoc test for pairwise comparisons. Statistical significance was defined as $*P < 0.05$, $**P < 0.01$, $***P < 0.001$, and $****P < 0.0001$. All statistical analyses were performed using GraphPad Prism 9 software. All experimentation was performed in triplicates (n = 3).

## 4. Results

### 4.1 Cytotoxicity and Anti-proliferation Activity

This study investigated the effects of ACA and cisplatin on SW620 cell viability and proliferation using the MTT and colony formation assays, respectively. SW620 cells were exposed to various concentrations of ACA (0 to 60 μM) and cisplatin (1.25 to 30 μM) for 24 to 72 hours. The results showed no significant cytotoxicity in the control group. In contrast, treatment with both ACA and cisplatin led to a significant reduction in cell viability in a dose- and time-dependent manner. The calculated $IC_{50}$ values for both compounds across the 24- to 72-hour treatment period, indicating growth inhibition, are presented in Figure 1 and Table 1.

To further evaluate the anti-proliferative potential of ACA, a colony formation assay was performed. The results showed a significant reduction in both the number of colonies formed and the percentage of colony-forming ability following treatment with cisplatin (6–12 μM) and ACA (10–20 μM). These findings indicate that both cisplatin and ACA possess significant anti-proliferative activity, as shown in Figure 2.

**Table 1** Cytotoxic activity ($IC_{50}$) of ACA and cisplatin in SW620 cells

| Cell lines | ACA (μM) | | | Cisplatin (μM) |
|---|---|---|---|---|
| | **$IC_{50}$ (24 hours)** | **$IC_{50}$ (48 hours)** | **$IC_{50}$ (72 hours)** | **$IC_{50}$ (48 hours)** |
| **SW620** | 53.15 ± 1.41*** | 50.97 ± 0.51*** | 40.23 ± 0.34 | 24.42 ± 1.10 |

Values are expressed as the mean ± standard error of the mean (SEM) of three independent experiments. The triple asterisks (***) denote a statistically significant difference with a p-value of less than 0.001.

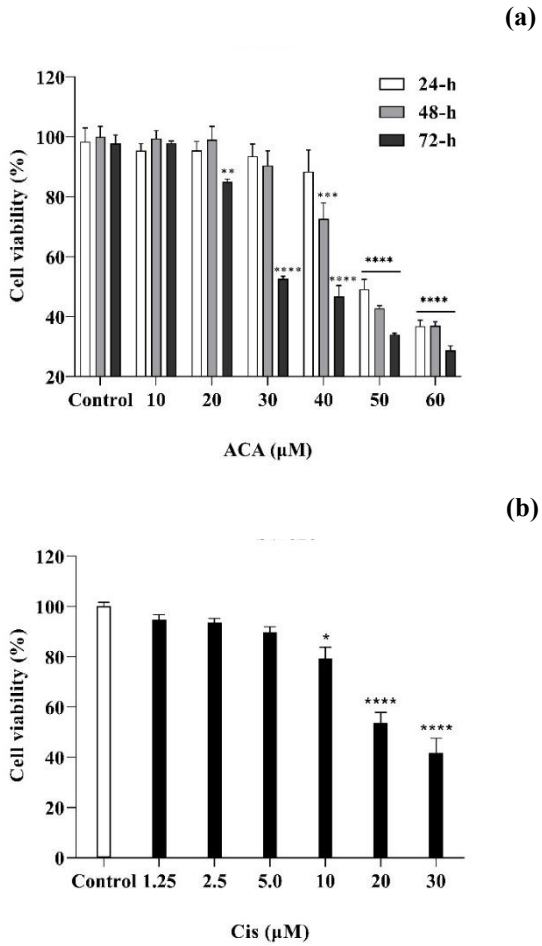


**Figure 1** Cytotoxic Effects of ACA and Cisplatin on SW620 Cells. (a) Half-maximal inhibitory concentration ($IC_{50}$) values of ACA in SW620 cells following 24, 48, and 72 hours of treatment. (b) Half-maximal inhibitory concentration ($IC_{50}$) value of cisplatin in SW620 cells following 48 hours of treatment. Statistically significant differences compared to the control group were determined by statistical analysis (*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$, and ****$P < 0.0001$). All experiments were performed in triplicate (n = 3)

### 4.2 Anti-migration Activity

The wound healing assay revealed that ACA treatment significantly inhibited SW620 cell migration and wound closure in a dose-dependent manner. Notably, a marked reduction in cell migration was observed at the higher concentration of ACA (20 μM) compared to the control group. These findings suggest that ACA effectively reduces the migratory capacity of SW620 cells, as shown in Figure 3.

### 4.3 Anti-invasion Activity

The effect of ACA on the invasive capacity of SW620 cells was evaluated using a Matrigel-coated transwell assay. The findings revealed a significant, dose-dependent reduction in the number of cells that invaded through the Matrigel barrier following treatment with ACA, as shown in Figure 4. These results strongly suggest that ACA possesses potent anti-invasive properties in colorectal cancer cells and warrant further investigation into its therapeutic potential.

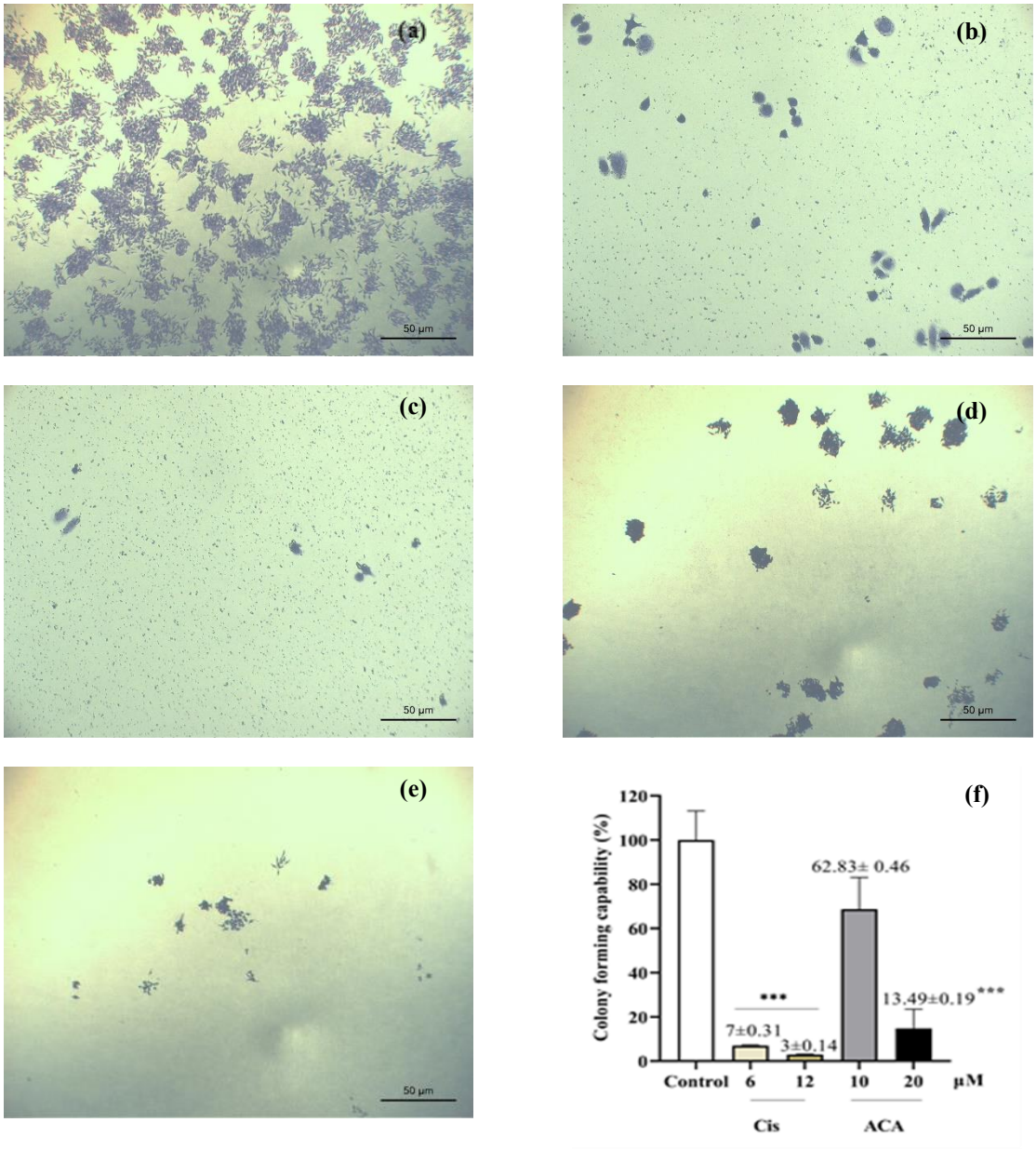


**Figure 2** Anti-proliferative effects of ACA and cisplatin on SW620 colorectal cancer cells determined by the colony-forming assay. Representative images of colonies formed under different treatment conditions: (a) untreated control, (b) cisplatin 6 µM, (c) cisplatin 12 µM, (d) ACA 10 µM, and (e) ACA 20 µM. Panel (f) shows the percentage of colony-forming ability after 7 days of treatment with cisplatin (6–12 µM) and ACA (10–20 µM). Data are presented as mean ± SEM, and statistical significance was determined relative to the control group (0.2% EtOH in RPMI). ***$p < 0.0001$. All experiments were conducted in triplicate (n = 3). Scale bars = 50 µm; images captured at 20× magnification

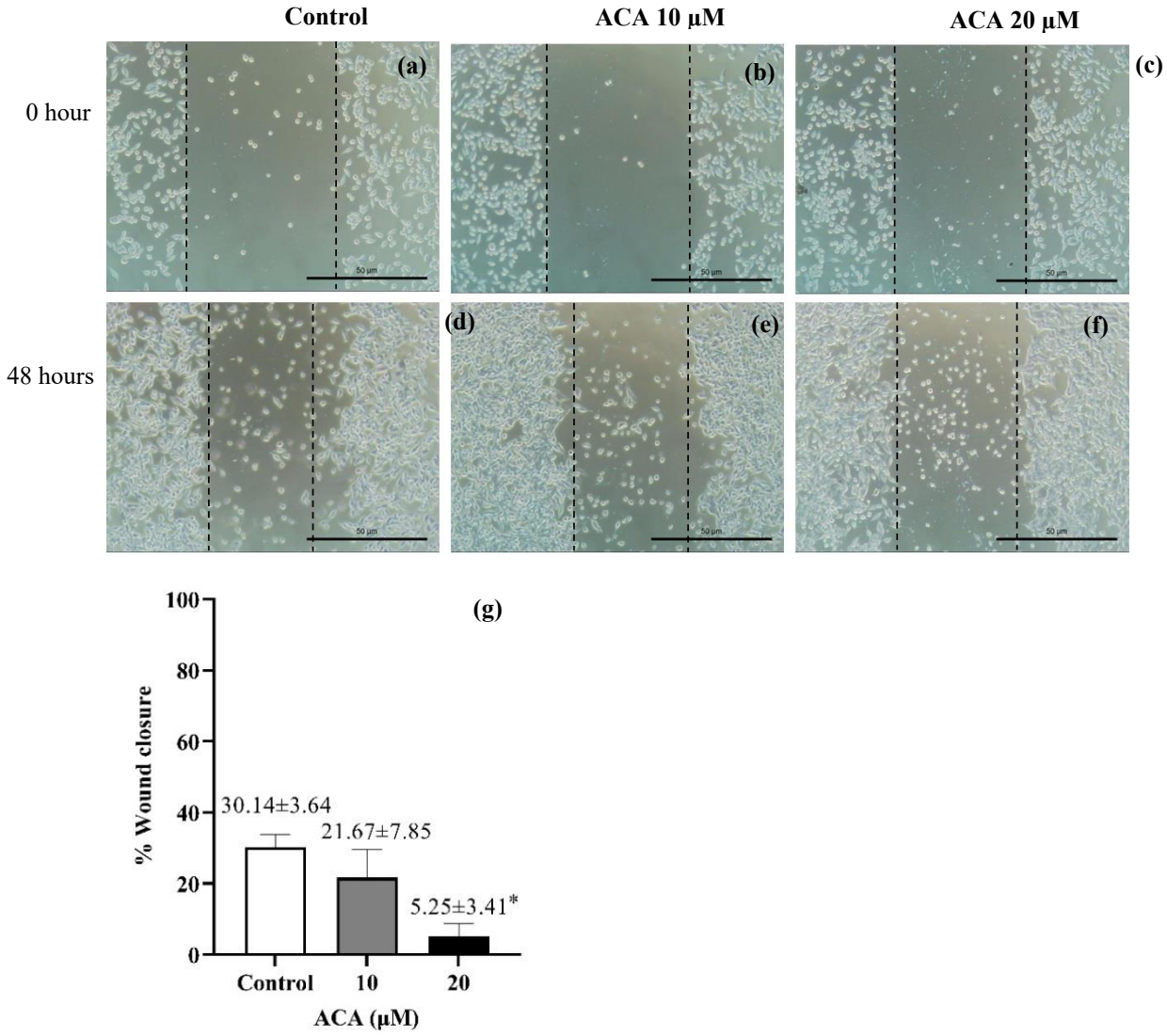


**Figure 3** ACA inhibits SW620 cell migration in a wound-healing assay. Representative micrographs showing wound closure under different treatment conditions: (a) untreated control, (b) ACA 10 µM, and (c) ACA 20 µM at 0 h; (d) untreated control, (e) ACA 10 µM, and (f) ACA 20 µM at 48 h. Panel (g) illustrates the percentage of wound closure after 48 h of treatment with ACA (10–20 µM). Data are presented as mean ± SEM. *$p < 0.05$ compared with the control group. All experiments were conducted in triplicate (n = 3). Scale bars = 50 µm; images captured at 40× magnification

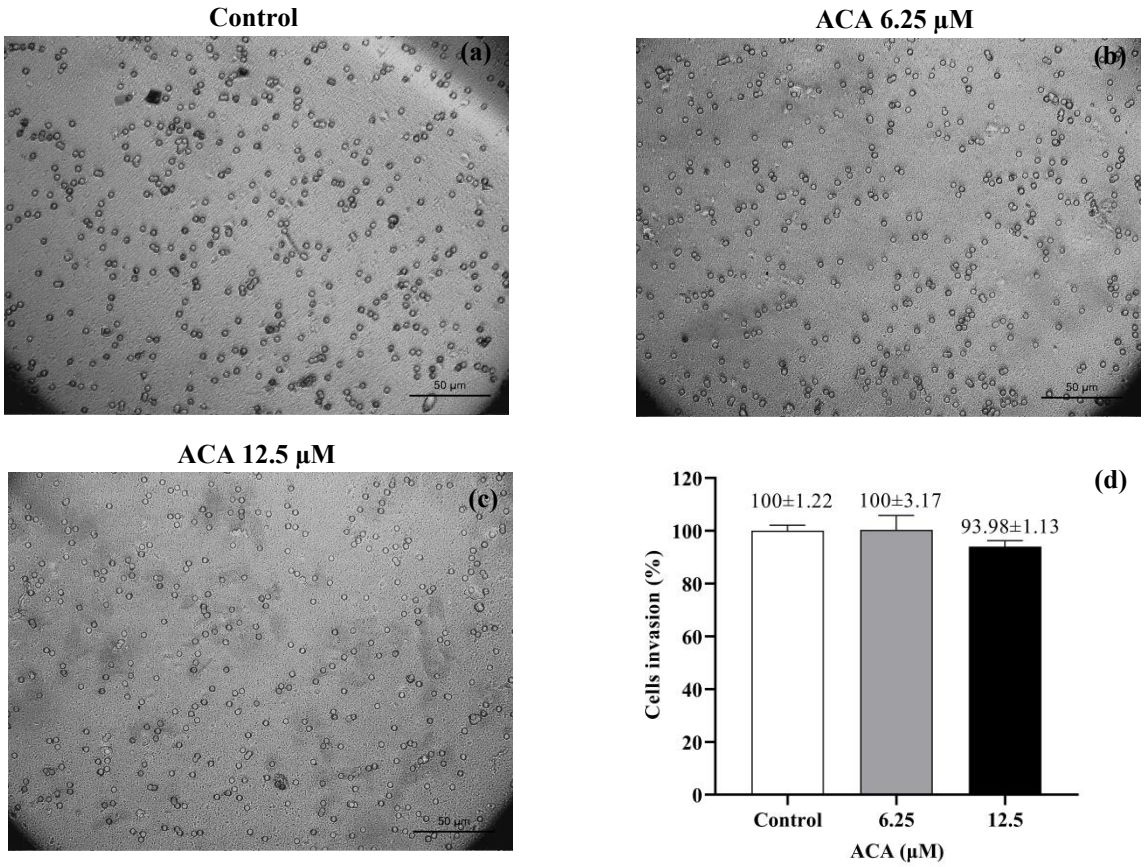


**Figure 4** Inhibition of SW620 cell invasion by ACA. Representative images of invaded cells: (a) untreated control, (b) treated with ACA 6.25 µM, (c) treated with ACA 12.5 µM, and (d) percentage of cell invasion after 48 hours of ACA exposure (6.25–12.5 µM). All experiments were performed in triplicate (n = 3). Scale bar = 50 µm (20× magnification)

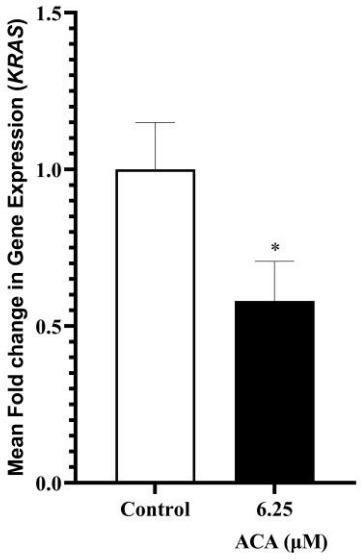


**Figure 5** *KRAS* gene expression in SW620 cells following ACA treatment. Relative *KRAS* mRNA levels in control and 6.25 µM ACA-treated SW620 cells after 24 hours, as determined by qRT-PCR. Expression was normalized to the *GAPDH* gene. $P < 0.05$ compared to the control group. All experiments were performed in triplicate (n = 3)

### 4.4 Regulation of Gene Expression in Cancer Cells

Given the involvement of *KRAS* in cell proliferation, we investigated whether the anti-proliferative activity of ACA was mediated through modulation of *KRAS* gene expression. Quantitative real-time PCR analysis revealed that treatment of SW620 cells with ACA at a concentration of 6.25 μM (a non-toxic dose selected based on MTT assay data) resulted in a significant downregulation of *KRAS* mRNA levels, as shown in Figure 5. This finding suggests that inhibition of *KRAS* expression may contribute to the observed anti-proliferative effects of ACA.

## 5. Discussion

Despite advancements in cancer therapies, cancer remains a leading cause of mortality, driving growing interest in natural compounds with potential anticancer properties, some of which have been historically used in traditional medicine. 1′-Acetoxychavicol acetate (ACA), a phenylpropanoid found in *Alpinia galanga* (L.), has a long history of use in culinary and traditional practices across diverse cultures and is increasingly recognized for its potential in cancer treatment (Xu et al., 2008; Kojima-Yuasa et al., 2020). Notably, studies have reported that ACA is safe in non-cancerous cells, with concentrations up to 80 μM showing no adverse effects on human mammary epithelial cells (Phuah et al., 2013).

Preclinical investigations have demonstrated that ACA can inhibit cell proliferation, induce apoptosis, and modulate various signaling pathways implicated in cancer progression. For instance, recent studies have shown significant reductions in colony formation and inhibition of migrationby ACA in human non-small cell lung cancer (Ketkomol et al., 2024). Recognizing the limited data on colorectal cancer, this study investigated the anticancer activities of ACA on SW620 cells. Our findings revealed that ACA exerted concentration-dependent cytotoxic and anti-proliferative effects in these cells, as evidenced by MTT and colony formation assays. Furthermore, evaluation of anti-metastatic potential through migration and invasion assays demonstrated that ACA significantly reduced SW620 cell migration and inhibited invasion.

Crucially, to gain insight into the underlying mechanisms, we examined the effect of ACA on the expression of *KRAS*, a key oncogene frequently mutated and overexpressed in colorectal cancer and known to drive cell proliferation and metastasis. Our quantitative real-time PCR analysis revealed a significant downregulation of *KRAS* mRNA levels in SW620 cells treated with a non-toxic concentration of 6.25 μM ACA for 24 hours, as illustrated in Figure 5. This reduction in *KRAS* expression at the mRNA level suggests that ACA may exert its anti-proliferative and anti-metastatic effects, at least in part, by targeting this critical signaling node.

*KRAS* encodes a small GTPase involved in numerous downstream pathways, including the MAPK and PI3K-AKT pathways, which are central to cell growth, differentiation, and survival (Bertotti et al., 2015; Aiello et al., 2019). Downregulation of *KRAS* can therefore have broad impacts on tumor cell behavior. Our finding aligns with the observed reductions in proliferation, migration, and invasion, suggesting a potential mechanistic link. *KRAS* is a particularly attractive therapeutic target due to its high prevalence in various malignancies, including colorectal cancer, where its dysregulation is often associated with aggressive disease and resistance to conventional therapies. While direct targeting of mutant *KRAS* has historically been challenging, modulating its upstream regulators or overall expression levels represents a promising alternative strategy.

Our data indicates that ACA may represent such a modulator, offering a potential avenue for therapeutic intervention in *KRAS*-driven colorectal cancers. These findings highlight the potential of ACA as a therapeutic agent against colorectal cancer, warranting further in-depth investigation into its mechanisms of action, including its effects on downstream *KRAS* signaling pathways, and its translational applications (Pradubyat et al., 2022).

## 6. Conclusion

The findings of this research strongly suggest that ACA exhibits significant anticancer activity against SW620 colorectal cancer cells. Future studies focused on preclinical validation and translational efforts are essential to determine the clinical potential of ACA in combating this prevalent disease.

## 7. List of Abbreviations

| | |
|---|---|
| ACA | 1'-acetoxychavicol acetate |
| ANOVA | analysis of variance |
| cDNA | complementary DNA |
| Cis | cisplatin |
| Ct | cycle threshold |
| DMSO | dimethyl sulfoxide |
| FBS | fetal bovine serum |
| *GAPDH* | Glyceraldehyde 3-phosphate dehydrogenase |

| | |
|---|---|
| HSD | Honestly Significant Difference |
| $IC_{50}$ | Half-maximal inhibitory concentration |
| *KRAS* | Kirsten rat sarcoma viral oncogene homolog |
| MTT | methylthiazolyldiphenyl-tetrazolium bromide |
| NCD | non-communicable disease |
| qRT-PCR | quantitative real-time PCR |
| RPMI | Roswell Park Memorial Institute-1640 |
| SEM | standard error of the mean |

## 8. Acknowledgements

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## 9. CRediT Statement

**Pataweekorn Ketkomol:** Conceptualization, methodology, writing original draft, formal analysis, validation, data curation.
**Thanapat Songsak:** Supervision, project administration, funding acquisition.
**Suchada Jongrungruangchok:** Investigation, visualization, funding acquisition, resources.
**Apirada Sucontphunt:** Visualization, resources.
**Fameera Madaka:** Methodology, formal analysis, data curation.
**Nalinee Pradubyat:** Conceptualization, investigation, formal analysis, validation, data curation, writing review & editing, project administration.

## 10. References

Aiello, P., Sharghi, M., Mansourkhani, S. M., Ardekan, A. P., Jouybari, L., Daraei, N., ... & Kooti, W. (2019). Medicinal plants in the prevention and treatment of colon cancer. *Oxidative Medicine and Cellular Longevity*, *2019*, Article 2075614. https://doi.org/10.1155/2019/2075614

Baradwaj, R. G., Rao, M. V., & Senthil Kumar, T. (2017). Novel purification of 1′S-1′-acetoxychavicol acetate from *Alpinia galanga* and its cytotoxic plus antiproliferative activity in colorectal adenocarcinoma cell line SW480. *Biomedicine & Pharmacotherapy*, *91*, 485-493. https://doi.org/10.1016/j.biopha.2017.04.114

Bertotti, A., & Sassi, F. (2015). Molecular pathways: Sensitivity and resistance to anti-EGFR antibodies. *Clinical Cancer Research*, *21*(15), 3377–3383. https://doi.org/10.1158/1078-0432.CCR-14-0848

Danaei, G., Vander Hoorn, S., Lopez, A. D., Murray, C. J., Ezzati, M., & Comparative Risk Assessment collaborating group (Cancers). (2005). Causes of cancer in the world: Comparative risk assessment of nine behavioural and environmental risk factors. *The Lancet*, *366*(9499), 1784–1793. https://doi.org/10.1016/S0140-6736(05)67725-2

Jongrungraungchok, S., Madaka, F., Wunnakup, T., Sudsai, T., Pongphaew, C., Songsak, T., & Pradubyat, N. (2023). *In vitro* antioxidant, anti-inflammatory, and anticancer activities of a mixture of Thai medicinal plants. *BMC Complementary Medicine and Therapies*, *23*, Article 43. https://doi.org/10.1186/s12906-023-03862-8

Horton, R. (2013). Non-communicable diseases: 2015 to 2025. *The Lancet*, *381*(9866), 509–510. https://doi.org/10.1016/S0140-6736(13)60100-2

Iacopetta, B. (2003). TP53 mutation in colorectal cancer. *Human Mutation*, *21*(3), 271–276. https://doi.org/10.1002/humu.10175

In, L. L., Arshad, N. M., Ibrahim, H., Azmi, M. N., Awang, K., & Nagoor, N. H. (2012). 1′-Acetoxychavicol acetate inhibits growth of human oral carcinoma xenograft in mice and potentiates cisplatin effect via proinflammatory microenvironment alterations. *BMC Complementary and Alternative Medicine*, *12*, Article 179. https://doi.org/10.1186/1472-6882-12-179

Ketkomol, P., Songsak, T., Jongrungruangchok, S., Madaka, F., & Pradubyat, N. (2024). The effect of 1′-acetoxychavicol acetate on A549 human non-small cell lung cancer. *Journal of Current Science and Technology*, *14*(2), Article 43. https://doi.org/10.59796/jcst.V14N2.2024.43

Kojima-Yuasa, A., & Matsui-Yuasa, I. (2020). Pharmacological effects of 1′-acetoxychavicol acetate, a major constituent in the rhizomes of *Alpinia galanga* and *Alpinia conchigera*. *Journal of Medicinal Food*, *23*(5), 465–475. https://doi.org/10.1089/jmf.2019.4490

Lamouille, S., Xu, J., & Derynck, R. (2014). Molecular mechanisms of epithelial-mesenchymal transition. *Nature Reviews Molecular Cell Biology*, *15*(3), 178–196. https://doi.org/10.1038/nrm3758

Mitry, E., Guiu, B., Cosconea, S., Jooste, V., Faivre, J., & Bouvier, A. M. (2010). Epidemiology, management and prognosis of colorectal cancer with lung metastases: A 30-year population-based study. *Gut*, *59*(10), 1383–1388. https://doi.org/10.1136/gut.2010.211557

Nelson, V., Sahoo, N., Sahu, M., Pullaiah, C., & Muralikrishna, K. (2020). In vitro anticancer activity of *Eclipta alba* whole plant extract on colon cancer cell HCT-116. *BMC Complementary Medicine and Therapies*, *20*(1), Article 355. https://doi.org/10.1186/s12906-020-03118-9

Phuah, N. H., In, L. L., Azmi, M. N., Ibrahim, H., Awang, K., & Nagoor, N. H. (2013). Alterations of microRNA expression patterns in human cervical carcinoma cells (Ca Ski) toward 1′S-1′-acetoxychavicol acetate and cisplatin. *Reproductive Sciences*, *20*(5), 567–578. https://doi.org/10.1177/1933719112459220

Pradubyat, N., Giannoudis, A., Elmetwali, T., Mahalapbutr, P., Palmieri, C., Mitrpant, C., & Ketchart, W. (2022). 1′-Acetoxychavicol acetate from *Alpinia galanga* represses proliferation and invasion, and induces apoptosis via HER2-signaling in endocrine-resistant breast cancer cells. *Planta Medica*, *88*(2), 163–178. https://doi.org/10.1055/a-1307-3997

Ray, S., & Pareek, A. (2023). A systematic review on using virtual assistance-based education and lifestyle interventions to prevent non-communicable diseases. *Journal of Current Science and Technology*, *13*(1), 118–135. https://doi.org/10.14456/jcst.2023.11

Thiha, P., & Sawasdipong, J. (2025). Efficacy and safety of sonic hedgehog inhibitors and PD-1 inhibitors in locally advanced basal cell carcinoma management: A systematic review and meta-analysis (2013–2023). *Journal of Current Science and Technology*, *15*(2), Article 110. https://doi.org/10.59796/jcst.V15N2.2025.110

Xu, S., Kojima-Yuasa, A., Azuma, H., Huang, X., Norikura, T., Kennedy, D. O., & Matsui-Yuasa, I. (2008). (1′S)-Acetoxychavicol acetate and its enantiomer inhibit tumor cell proliferation via different mechanisms. *Chemico-Biological Interactions*, *172*(3), 216–223. https://doi.org/10.1016/j.cbi.2008.01.002

Xu, X. Y., Zhao, C. N., Cao, S. Y., Tang, G. Y., Gan, R. Y., & Li, H. B. (2020). Effects and mechanisms of tea for the prevention and management of cancers: An updated review. *Critical Reviews in Food Science and Nutrition*, *60*(10), 1693–1705. https://doi.org/10.1080/10408398.2019.1588223